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Hard Tissue Lesions Associated with Malnutrition

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HARD TISSUE LESIONS ASSOCIATED WITH MALNUTRITION

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The hard tissues, bone, cartilage and teeth, are very sensitive to improper nutrition and react very quickly to even minor variations in the ration. Not only are these reactions distinct but often they appear early in the course of an illness. Frequently they are an indelible imprint of the experience and persist for the entire life of the animal.

There is a general tendency to regard hard tissue diseases as a manifestation of a deficiency of some substance in the ration. On the contrary, the concept of **nutritional balance** is the key to diagnosis and rational treatment of hard tissue diseases. The point which is usually overlooked is that an excess of any ingredient in the ration is just as important as a deficiency in the production of a nutritional disease. The same lesion may be produced by an excessive amount of a certain substance in the ration as will be produced by an inadequate amount.

Nutritional balance can best be illustrated with the teeter-totter. If an excess of one substance appears in the ration then there must also be a corresponding increase in other ingredients if nutritional balance is to be maintained. The teeter-totter moves up and down during the lives of our animals but as long as it continues to fluctuate constantly a nutritional balance will be maintained.

To this first factor, nutritional balance, let us add the second important factor in nutritional disease and that is **rate of growth.** This factor is especially important in the diseases of hard tissue, and is the key to success or failure in the production of lesions in hard tissue research. The faster the rate of growth the greater is the probability that a bone disease will appear if the nutritional balance is not correct. In contrast, a slow rate of growth decreases the likelihood that a hard tissue disease will develop. Therefore, it is the rate of growth which determines why a group of animals in one experiment or on one farm develops a bone disease while another group of animals in another experiment or on another farm does not develop the disease even though the ration, management, and environment are similar.

This second factor, rate of growth, is best illustrated by the height of the fulcrum in the diagrammatic teeter-totter. If the fulcrum is in the center of the plane of balance and of the proper height, then normal fluctuation can occur. However, if the rate of growth is increased or rather the height of the fulcrum is greater than normal, then any variation in balance produces a greater excursion of the plane of

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balance and the teeter-totter tips excessively to the side of the greater ration imbalance and a hard tissue disease is the result. A slow rate of growth can be depicted by lowering the height of the fulcrum which in turn reduces the possible magnitude of the excursion of the plane of balance. If the rate of growth is greatly reduced the fulcrum becomes so low that extensive excursions of the plane of balance are limited or impossible and therefore a hard tissue disease will probably not occur.

The third factor which exerts an influence on the hard tissues is the **intrinsic causes of disease** (genus, breed, race, family, color, sex, age, and idiosyncrasy) over which the individual himself has no control. Large breeds of dogs (Saint Bernard and Great Dane) have a greater incidence of bone disease than do the smaller breeds. The Filipino pony is less likely to develop bone disease as the result of a deficiency of phosphorus than is the Thoroughbred. Rickets in the human being occurs in the young and is more frequently observed in males than in females.

The importance of the intrinsic causes of disease in the production of hard tissue lesions can be illustrated by the position of the fulcrum along the plane of balance. If the intrinsic causes are compatible with normal growth then the diagrammatic fulcrum can be placed in the center of the plane of balance. However, if an intrinsic cause of disease exerts an incompatible influence on growth then the fulcrum is shifted to some position other than the center of balance. Then, unless a compensating adjustment is made in the ration, the probability exists that a hard tissue disease will result.

It is a fallacy to believe that each nutritional defect in the ration will produce a pathognomonic lesion. The entire basis of pathology is that tissue reacts in a limited number of ways and a large number of etiologic agents will produce an identical tissue reaction. As a result, instead of trying to attach pathognomonic importance to each hard tissue alteration, the causes of hard tissue disease should be arranged into groups which have similar lesions. Once the basic lesions of the group are recognized, it is then comparatively easy to decide the probable cause or causes of the existing hard tissue disease.

Lesion A. Excessive amounts of osteoid tissue produced by osteoblasts.

The presence of excessive amounts of osteoid tissue indicates there is a normal or increased rate of osteoblastic activity together with an inadequate mineralization of the newly formed tissue. It is the type of reaction observed when inadequate amounts of vitamins D and minerals, especially calcium and phosphorus, are present in the ration. It represents the failure of the individual to mineralize the osteoid tissue thereby converting it into osseous tissue. This is the type of reaction observed in classical rickets in the human being, where failure of mineralization is attributed primarily to a deficiency of vitamins D.

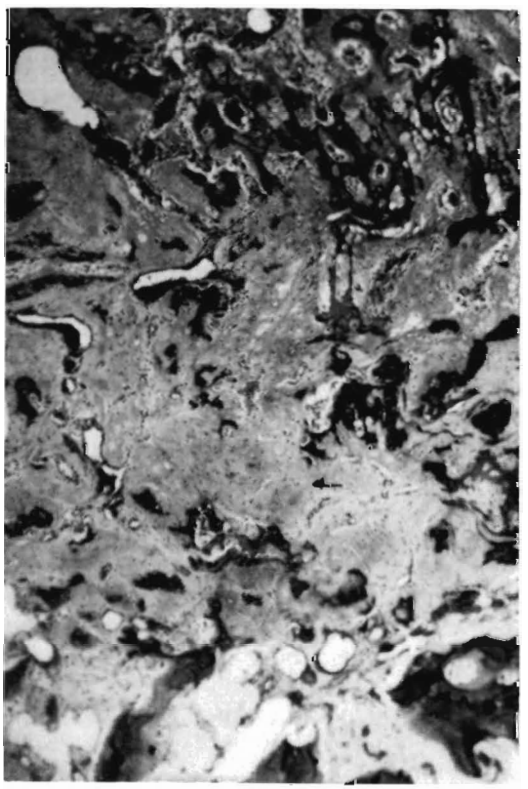

Fig. 1. The arrow indicates the excessive amounts of osteoid tissue at the costochondral junction. Note how the tisuse has been compressed, twisted, and distorted. x100.

Fig 2. An island of cartilage which is undergoing metaplasia. The arrow indicates the new osteoid tissue. x100.

Classical rickets occurs only in primates whose bone development is slow. In other species of animals, bone growth, at the time of birth, has already passed that period of development in which classical rickets can occur.

A form of rickets can be produced in the rat and chick. However, to accomplish this the vitamins D, calcium, phosphorus, and nutritive constituents of the ration must be present in very definite amounts, and a rapid rate of growth must be obtained if excessive osteoid tissue is to be produced. The only reason a disease simulating classical rickets can be produced in the rat and chick is that the young of these animals have a comparatively poorly developed skeleton at birth, and on an experimental ration, if they can be made to maintain a rapid rate of growth, they may develop an osteodystrophy.

The appearance of excessive amounts of osteoid tissue in other species of animals is seldom observed, and when it does appear it does so under very unusual conditions and involves only certain species of animals. A deficiency of vitamins D alone will probably not produce classical rickets in domesticated animals. To produce excessive amounts of osteoid tissue in domesticated animals there must also be in mineral imbalance and the animals during the experimental procedure must maintain a rapid rate of growth (Fig. 1). If a rapid rate of growth cannot be maintained, osteoblastic activity is also suppressed and osteoid tissue is not produced in excess. Without excessive amount of osteoid tissue a diagnosis of rickets cannot be made.

Lesion B. Excessive amounts of osteoid tissue produced by metaplasia of cartilage and connective tissue.

Metaplasia of cartilage into osteoid tissue is frequently observed in bone disease and is the attempt of the tissues to compensate for the impaired osteoblastic endochondral ossification (Fig. 2 and 3). It is

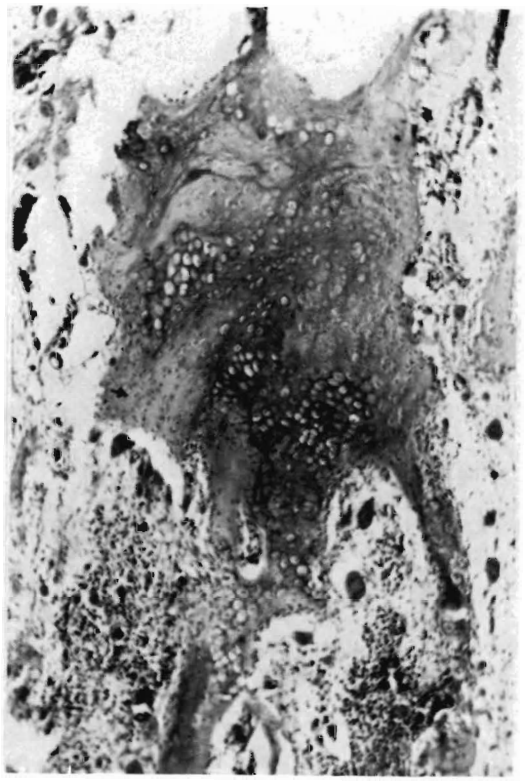

Fig: 3. Conversion of cartilage into osteoid tissue. x100.

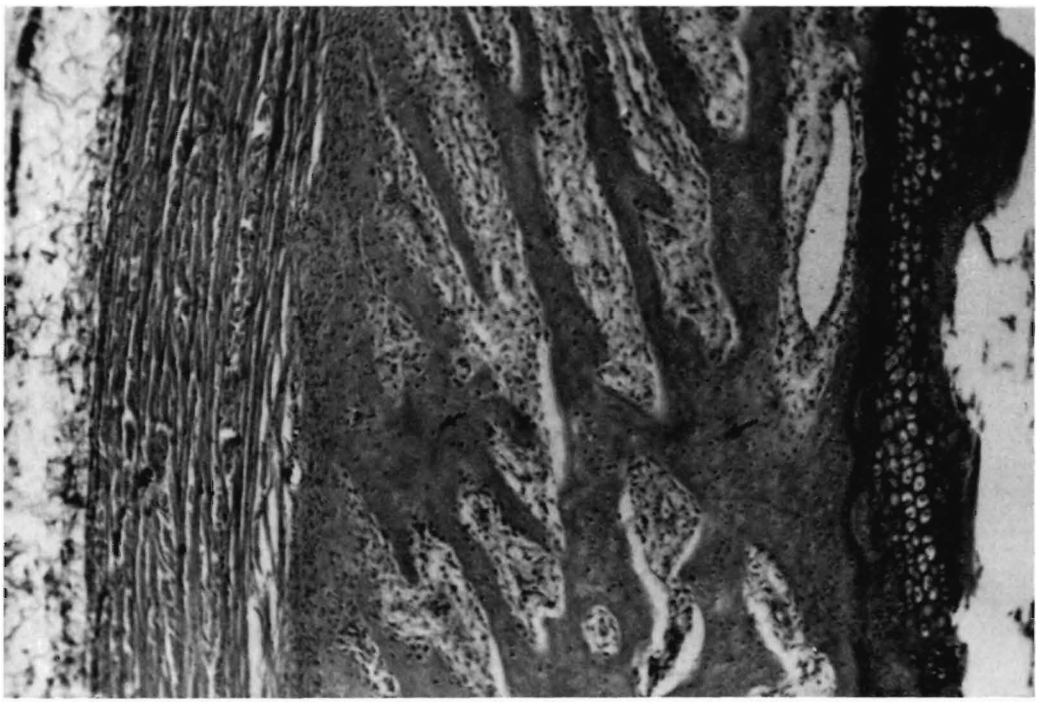

Fig. 4. Metaplasia of white fibrous connective tissue in the periosteum into osteoid tissue. The arrows indicate the osteoid tissue. x100.

the most frequent cause of excessive osteoid tissue in turkeys and chickens and is the lesion observed in the so-called rickets in poultry. **It is not a diagnostic lesion for a deficiency of vitamins D in the ration.** Instead, it is an indication of an osteopathy which may be due to a variety of causes. It is usually due to mineral imbalances (calcium, phosphorus, magnesium, and iron) in the ration and seldom, if ever, is due to a deficiency of vitamins D alone.

The white fibrous connective tissue in the bone marrow and periosteum, especially in the vicinity of the epiphyseal plate, may undergo metaplasia and change into osteoid tissue (Fig. 4). The connective tissue in this region becomes excessive when an osteopathy is present. This hyperplasia of connective tissue is the reaction of the bone to the stresses and strains placed upon it when it lacks the strength and rigidity required for normal body support and movement. It is especially prominent in the proximal ends of the ribs and is the lesion which is erroneously used as an indication that a deficiency of vitamins D is present (Fig. 5).

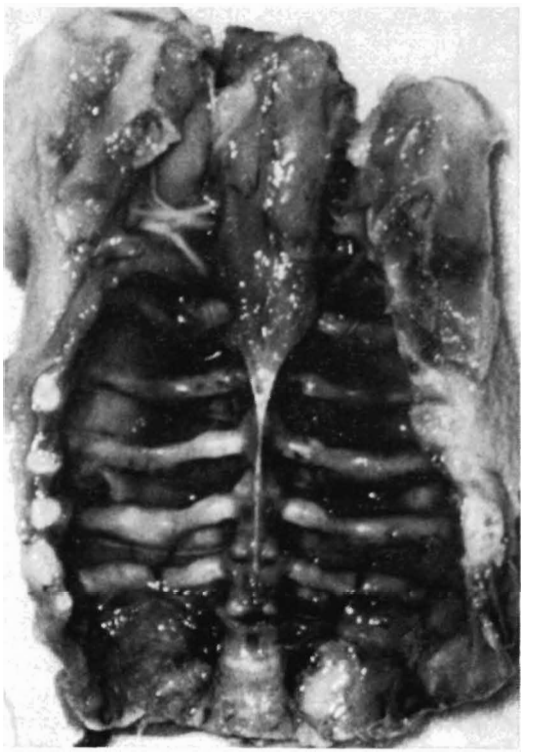

Fig. 5. Note the beading and distortion of the ribs. The alterations in the ribs were caused by an excessive amount of magnesium in the ration.

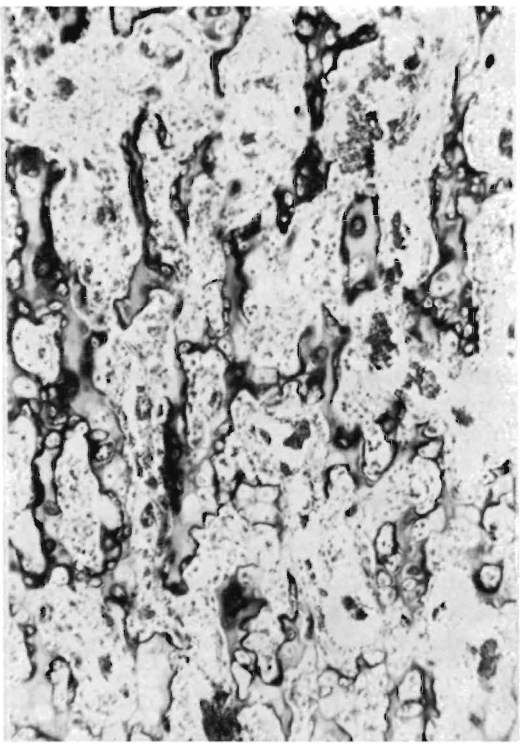

Fig. 6. Inadequate amounts of osteoid tissue. Note the cartilagenous trabeculae are not enclosed with osteoid tissue and that osteoblasts are not present. x100.

Lesion C Inadequate amounts of osteoid tissue.

In the normal development of bone, osteoid tissue must be present if osseous tissue is to be formed. If osteoid tissue is not produced bone growth does not occur. Osteoid tissue is produced by the osteoblast. This cell is extremely sensitive to even minor changes in the nutrition and health of the animal. Inadequacies in fat, carbohydrate, or protein, as occurs in starvation, are immediately reflected by a suppression or cessation of osteoblastic activity (Fig. 6). Stunting of the animal is the result.

The osteoblast is also very sensitive to the presence of vitamin A. Apparently vitamin A is very closely associated with the metabolic activity of this cell. A deficiency of vitamin A results in hypoplasia or atrophy of the osteoblast. When this occurs bone ceases to grow. A similar reaction occurs when too much vitamin A is present. An excessive amount is toxic to the osteoblast. As a result, osteoid tissue is not produced and normal bone growth does not occur. This is one of the dangers associated with the adding of excessive amounts of vitamin A to the ration, especially when animals are feeding on luxuriant pasture. Again the biological teeter-totter of balance is exerting its influence and neither an excess or a deficiency of vitamin A can exist if normal bone growth is to be obtained.

The osteoblast is also very sensitive to the action of bacteria and viruses. Hog cholera, either the vaccination for hog cholera or the natural disease, is an excellent example of this. The hog cholera virus causes hypoplasia, atrophy, and even death of the osteoblast. The swine influenza, canine distemper, avian encephalomyelitis virus, as well as other viruses, will produce a similar reaction in the osteoblast. When this occurs bone growth ceases. The effects of these infectious agents on the osteoblast and the resulting changes in bone have led to many erroneous diagnoses of rickets in both swine and poultry.

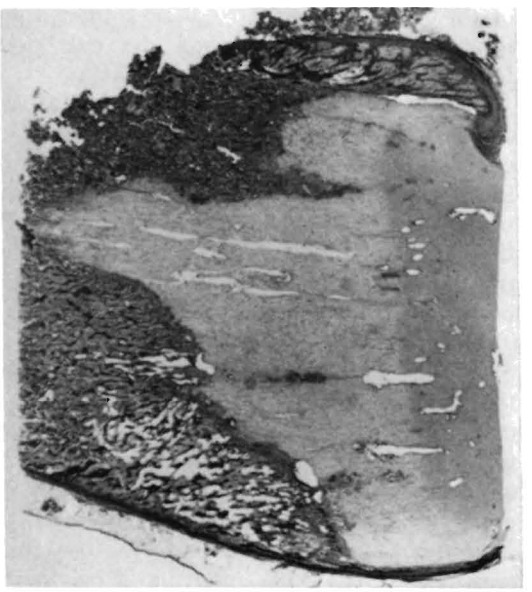

Fig. 7. The epiphyseal line is irregular and long tongues of cartilage persist in the diaphysis. x4.5.

Lesion D. Excessive amounts of cartilage as manifested by excessvely broad and wide epiphyseal plates.

This is an indication that the cartilage is not being invaded by the capillaries from the bone marrow and that endochondral ossification is not occurring. The zones of cartilage columns and vesicles are excessively broad. The epiphyseal line becomes very undulating and irregular, and long tongues of cartilage persist in the diaphysis (Fig. 7). The excessive amount of cartilage is due to the inability of the capillaries to invade the cell walls of the apparently mature cartilage cells of the vesicular zones of cartilage.

The inability of the capillaries to invade the cartilage cells is, at times, due to a deficiency or an excess of vitamin A. The endothelial cells do not show the active hyperplasia they normally do. Even if invasion of the cartilage lacunae does occur, metaplasia of the endothelial cells into osteoblasts does not occur or is suppressed and osteoid tissue is not formed.

The endothelial cell is also very sensitive to various viral diseases. The hog cholera virus is particularly injurious to endothelial tissue and when the endothelial cells are injured by either the attenuated virus of the vaccine or the virulent virus of the natural disease, the endothelial bud of the invading capillary is no longer able to penetrate the walls of the cartilage lacunae and produce osteoblasts.

Faulty nutrition or chronic debilitating disease also prevents capillary invasion of cartilage. The endothelial cells of the capillary bud are very sensitive to malnutrition, soon lose their normal vigor, and then are no longer able to invade the mature vesicular cartilage cells. Cartilage is not as easily injured by faulty nutrition, infectious diseases, or chronic debilitating diseases as is the osteoblast and the endothelial cells of the capillaries. As a result, cartilage continues to grow at a normal or near normal rate even though there is a suppression of the rate of conversion of cartilage into bone. Because of this discrepancy between cartilage formation and conversion, excessive amounts of cartilage are present. This causes the epiphyseal region of the bone to be unusually large in proportion to the underdeveloped diaphysis. The enlargement of the ends of the bones as the result of the excessive amounts of cartilage, which is mistaken for osteoid tissue, often leads to an erroneous diagnosis of rickets.

A deficiency of vitamin C may produce a similar reaction. When a deficiency of vitamin C is present there is, in addition to the suppressed activity of the endothelial cells and the osteoblasts, an increased fragility of the enodthelial cells of the capillaries which results in hemorrhage. If hemorrhage occurs along the epiphyseal line, large lakes of blood appear in the area and the capillaries which are present in this region are obliterated. Since the capillaries are no longer present, no invasion of cartilage occurs in this area, a long tongue of mature cartilage may then persist in the diaphysis, and no bone is produced in this area. Hemorrhage from traumatic injury or other diseases, such as hemorrhagic disease in the chicken, will cause a similar interferernce with bone growth.

Fig. 8. Twisted and compressed cartilagenous, osteoid, and osseous trabeculae. x100.

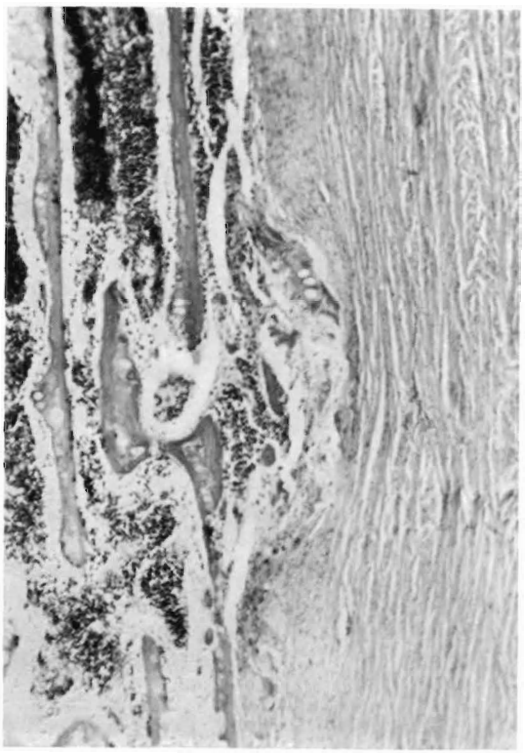

Fig. 9. Fracture of the trabeculae adjacent to the periosteum. x100.

Lesion E. Twisted, compressed, distorted, and fractured cartilagenous, osseous, and osteoid trabeculae.

This is an indication that faulty mineralization of cartilagenous, osteoid, and osseous tissue is present and that these structures do not have sufficient rigidity to support the weight of the animal and the stresses and strains placed upon it (Figs. 8, 9, and 10).

This lesion may be due to a deficiency of vitamins D which are necessary for the most efficient use of mineral by the body. In most of our domesticated animals a deficiency of vitamins D alone will not produce the bone lesion. It is associated with a deficiency or an excess of some mineral (calcium, phosphorus, iron, magnesium, and sulfur) in the ration.

Because of the distortion which results at the ends of the bones, this lesion is frequently called rickets. In the midwest, particularly in horses, feeder cattle, swine, and poultry, it is most frequently caused by a deficiency of calcium in the ration. In those areas of the country, such as the Southwest, where a phosphorus deficiency

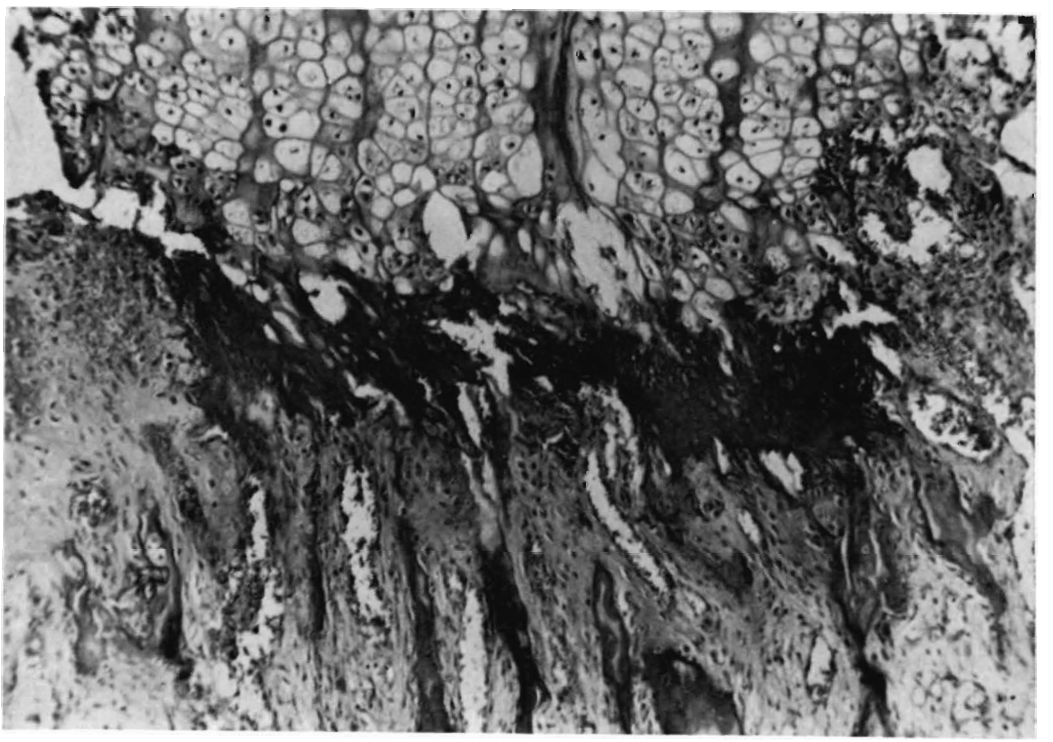

Fig. 10. Fracture at the junction of the prolifer :ting cartilage and the diaphysis. Normal bone growth can no longer occur in this area. x100.

in the soil exists, the same lesion will occur because of the imbalance of the calcium-phosphorus ratio.

Lesion F. A decrease in the number and size of the osseous trabeculae.

This lesion (Fig. 11) is due to two factors. First, it is associated with a suppressed activity of the osteobalsts in which adequate amounts of osteoid tissue are not produced. Without the osteoid tissue, osseous trabeculae are not formed. The causes of inadequate amounts of osteoid tissue have been discussed under Lesion B.

Secondly, it is associated with an imbalance of mineral in the ration in which the mineral stores in the bone are removed to supply the needs of metabolism and as a result of this withdrawal, atrophy of the osseous trabeculae occurs. This is commonly observed in the bones of the laying hen since progressive osteomalacia occurs during the egg laying period unless adequate amounts of mineral are added to the ration.

Fig. 11. Decrease in the number and size of the osseous trabeculae. x100.

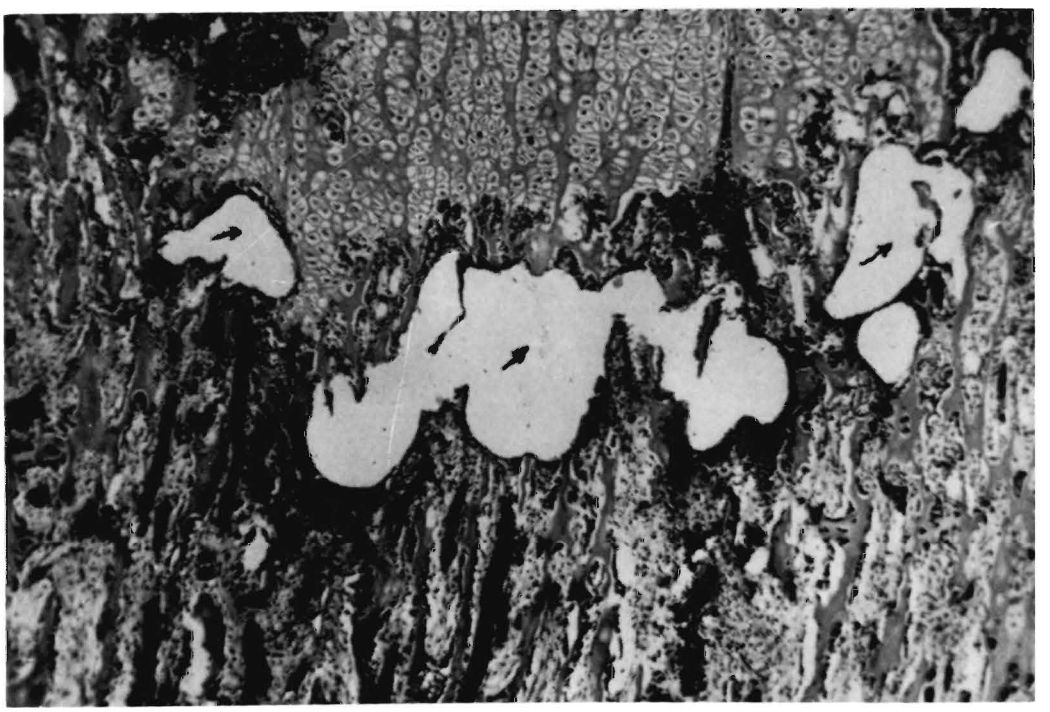

Fig. 12. The spaces indicated by the arrows are areas where hemorrhage has occurred and blood has been removed. Note the cartilagenous and osseous trabeculae are no longer continous in the area of hemorrhage. Imperfect bone development is the result. x100.

Lesion G. Hypoplasia of the bone marrow.

Hypoplasia and even aplasia of the bone marrow occurs during a deficiency of vitamin A. It may also occur during gross malnutrition, chronic debilitating diseases, and in some toxicoses. It is also a manifestation of some viral diseases such as hog cholera. (See Figs. 6 and 11)

Lesion H. Hemorrhage in the bone marrow and periosseous structures.

Hemorrhage is observed in scurvy and is best demonstrated in the guinea pig. It is found to be present in hemorrhagic disease in the chicken, sulphide and sulphite poisoning, septicemic disease such as hog cholera, and when traumatic injury to bone ocurs. The hemorrhage may seriously interfere with the growth of bone and is particularly injurious if it occurs along the epiphyseal plate where active bone growth is taking place. (Figs. 12 and 13).

Lesion I. Periostitis.

The periostitis associated with ration imbalances occurs at those points where tendons and ligaments are inserted into bone. If the bone is not of normal strength it is not able to withstand the stresses and strains placed upon it by the ligaments and tendons. Irritation and even minute fractures occur in the areas of attachment of these structures and periostitis is the result (Fig. 9). It is particularly common when calcium, phosphorus, magnesium, iron, copepr, and vitamins A, C, and D imbalances are present.

Lesion J. Thrombosis of blood vessels.

This lesion is most frequently observed in the epiphyseal plate (Fig. 14). It is a common lesion when adequate mineralization has not taken place. This lesion is seen more frequently in the turkey than in other species. The character and location of the lesion suggests that the inadequately mineralized cartilagenous and osseous structures are compressed by the weight and movement of the animal and crush the soft structures, such as blood vessels, which they contain. The injury to the endothelium of the blood vessel results in thrombosis.

Fig. 13. Hemorrhage in the periosteum. x100.

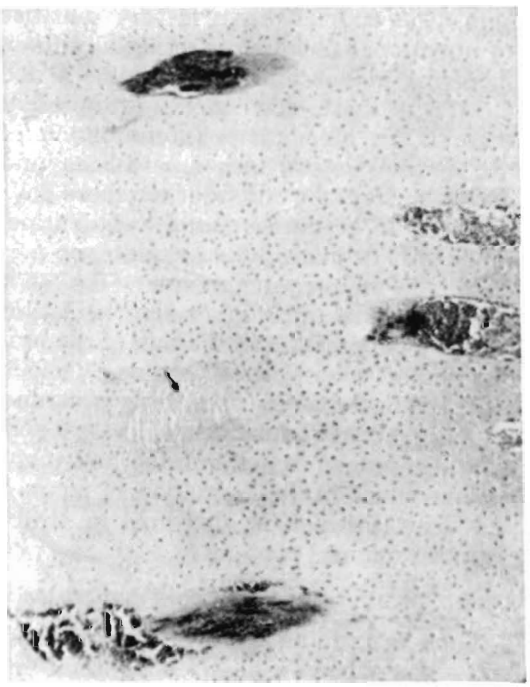

Fig. 14. Thrombi are present in three blood vessels. The arrow indicates an area of necrosis. x100.

Thrombosis of the blood vessels is very frequently observed when septicemic diseases (hog cholera, staphylococcosis, and salmonellosis) are present. It is also a common finding when chronic respiratory disease is present in poultry and probably explains part of the lameness observed in flocks affected with this disease.

Lesion K. Degeneration and necrosis of cartilage.

This lesion is most frequently observed when thrombosis of the nutrient vessels, as described in Lesion J, has occurred. The cartilage in the immediate vicinity of the thrombosed vessel shows degenerative alterations. At times, focal areas of liquefactive necrosis will be observed in the zones of columns and resting cells. These appear as small cyst-like structures (Fig. 15). The degeneration and necrosis is a manifestation of the infarction which occurs when the blood supply is destroyed.

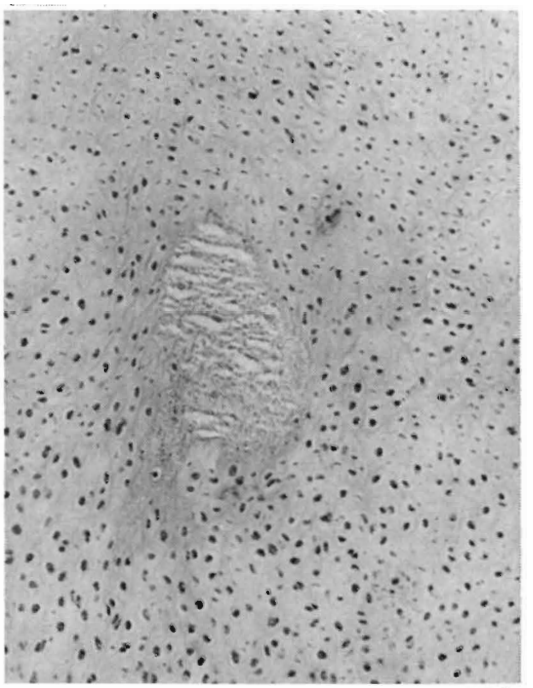

Fig. 15. A focal area of necrosis in the epiphyseal cartilage near a thrombosed vessel. x440.

Lesion L. Hypoplasia of enamel and dentine.

Since teeth originate from epithelium it is logical that dental defects will occur in those nutritional diseases which affect the skin. The enamel organ is very sensitive to nutritional imbalances, especially those involving magnesium and vitamins A and C. When these deficiencies are present there is an incomplete differentiation of the enamel organ into ameloblasts and odontoblasts. As a consequence these cells may fail to produce enamel and dentine, produce it in inadequate amounts, or produce it in an irregular manner (Fig. 16).

General malnutrition (starvation) also causes a suppression in the rate of the production of enamel and dentine. Various viral and bacterial diseases, such as canine distemper, cause hypoplasia of enamel and dentine. Imbalances of fluorine and magnesium result in the formation of enamel and dentine which is abnormal in structure and mineral content.

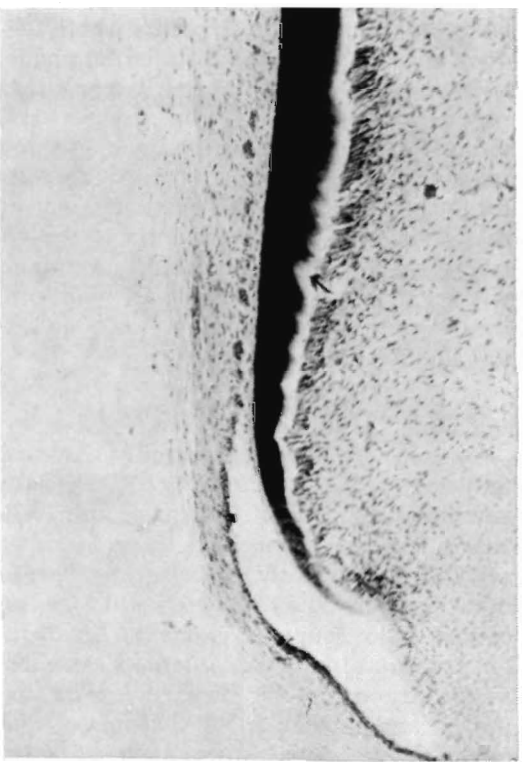

Fig. 16. Hypoplasia of dentine. Note the fragile structure of the root of the tooth and the irregular manner in which the dentine (arrow) has been formed. x100.

Lesion M. Peridontal osteoid hyperplasia

When examining teeth the peridontal bone may show excessive amounts of osteoid tissue (Fig. 17). This lesion is due to a deficiency of vitamins D and an im-

Fig. 17. Peridontal osteoid hyperplasia. Note how the root of the tooth has been distorted and compressed as the result of inadequate mineralization (compare with Fig. 16). The arrow indicates the osteoid hyperplasia. x100.

balance of minerals. Excessive amounts of osteoid tissue has been prevously discussed under Lesion A.

Lesion N. Disproportionate rates of body growth as compared with osseous growth.

By selection, diet, and environment an animal can be produced which has an amazingly rapid rate of growth. The most critical tissue in this rapidly growing animal is the skeleton. Apparently the osseous tissue has considerable difficulty in developing rapidly enough to support the ever increasing weight of the body. This is especially true in the pig which normally has a very small skeleton. It is also well known that the more rapidly bone grows the more critical becomes its nutritional requirements if the biological teeter-totter is to be kept in balance. As a result bone disease is very common in rapidly growing animals. — end

Veterinary Medicine in the U.S.

Veterinarians in 1960 had a higher net income before taxes than in 1955, still work comparatively long hours per week, and are moving away from self-employment, a preliminary economic study recently reported.

The survey was prepared by the AVMA Council on Veterinary Services and is reported in the current (Feb. 15) issue of the Journal of the American Veterinary Medical Association (pages 366-68). Its 1960 data can be compared with the Council's 1950 and 1955 surveys.

Average net income before taxes was $12,345 in 1960, 15 per cent higher than 1955 ($10,694) and 65 per cent higher than 1950 ($7,374). Income was highest in the Western states (California, Colorado, Idaho, Montana, Nevada, Oregon, Utah, Washington and Wyoming) and lowest in the Southwestern states (Arizona, Arkansas, New Mexico, Oklahoma, and Texas). The Western average was $13,723 and the Southwestern average $10,429.

More than half of the veterinarians reported work weeks in excess of 60 hours. As in 1955, most placed themselves in the 60–69 hours-per-week category. Fewer veterinarians, however, reported working 70 or more hours a week than did in 1955.

Veterinarians answering the survey (3,281 of 9,256, or 36 per cent, returned usable answers) said more of their income is coming from small animal practice and less from large animals. Swine practice income (12 percent in 1955 and 11 percent in 1960) and horse practice income (four percent both years) was static, but cattle practice income fell from 34 percent in 1955 to 26 percent in 1960. Small animal practice income rose from 45 percent in 1955 to 55 percent in 1960.

Whereas almost eight out of ten veterinarians (78 percent) were self-employed in 1955, only seven out of ten (69.7 percent) were in this category in 1960. In 1960, 15.9 percent said they were in partnership and 14.2 percent were salaried (total 30.1 percent); in 1955, 22 percent were in partnership or salaried.